

To Whom it May Concern:

September 25, 2017

Subject: Tort Claim No. 13648, Federal District Court , Sherman Texas

My name is Cynthia A. Vann, I am the widow of Navy Veteran, Philip D. Vann that I had filed Tort Claim No. 13648 for failure to diagnose and treat and wrongful death. I had filed an appeal with the General Counsel with the Department of Veterans Affairs in Washington. I was advised at that time there would be a reconsideration of my Claim. I was advised that the Attorney General's regulations provide their agency a 6-month period during which time my right to file suit is suspended. Thereafter, if no decision has been made, I may then either deem the matter denied by filing suit in Federal district court, or continue to wait an agency decision.

Their 6-month time frame was up April 2017, I have never received any correspondence saying my claim was denied. I am offended at such a mannerism of not even being notified of a decision. This is a disgrace to ignore me as this proves to me a widow of a veteran is a nobody to this system. I have decided after months of disappointment to file suit as the legal system allows, within 6 months from April 2017 time frame.

This letter has been sent certified mail, so I will be in receipt of the court receiving this letter with the date.

I am notifying this court today that I am filing a suit with this Federal district court, I am asking respectfully that you would give me the respect to look at the evidence that the Dallas VA Hospital denied my beloved husband medical treatment for. They knew for eight consecutive years he had an Agent Orange disease that not only did the VA Rating Board deny him Agent Orange Benefits for in 2011, but they also knew year after year of this heart disease that caused multiple health problems. It doesn't take a rocket scientist to understand that if a heart has blockage as his autopsy proves it is going to cause other major health problems.

My beloved husband had been denied Agent Orange compensation even though it was documented in his Dallas VA hospital medical records, one being radiology reports that were passed on to his lung Doctor time and time. Doctor or Doctors never seemed to care to get him help, they just let every evidence of life threatening heart disease to be overlooked as it meant nothing to them. He was never referred to a cardiologist. Doctors reading these radiology had an obligation to treat him, they never did anything for him and that is absolute negligence, they failed to diagnose him and treat him knowing he had heart blockage that continued to get worst and worst and became his demise. They

were quick to diagnose him with executive dementia, which he never had, he was gasping to breathe, no oxygen was getting to his brain. His lungs got scarred from multiple bouts of pneumonia because of his heart not pumping blood to his lungs to heal them, the scarring caused fibrosis. The VA will LIE and tell you different. He was my husband, I was his caregiver, we were married for 28 years. He NEVER had shortness of breath until his heart became a problem in 2008, when he went into A-Fib and they never did anything but put him ICU for 4 days, there were never any follow ups to a cardiologist, they knowingly let his heart get worst, knowing it would eventually kill him. We can't live without our heart.

His autopsy said he had Atherosclerotic, CardioVascular Disease, you do not get this over night, he had it for years and the Dallas VA hid it from him and me his caregiver and did nothing to help him, a simple stent could have cleared his blockage. He was having what they thought were seizures when in fact his autopsy said were strokes, which was from heart disease, and nothing was done. The Dallas VA knew he had A-Fib but he NEVER had a cardiologist, he was never seen by one other than the one time in their ICU for an A-Fib attack. I believe because he was 100% disabled from PTSD they just let him die, one less veteran to have to deal with and compensate. He was my husband, my children's father, he was a grandfather, he was NOTHING to the Dallas VA but another sick veteran to get off their books.

I am 62 years old, I'm grateful to be in excellent health, I am working full time, but his death has left me with no savings, had he lived longer and his heart given the proper treatment things could have been different. I was his caregiver, I kept him immaculate, he was safe, he was a fighter until the end. He was an intelligent man, who went to war right out of highschool top of his class, he wanted to serve his country, he came back a broken young man. He like all of our Vietnam Veterans were never welcomed home, he suffered from survivors guilt, he was a kind man, wanting to help others but couldn't overcome his own battle with PTSD. I loved him, comforted him, told him it was ok to cry, to never to be ashamed of what he was battling. He had a funeral that of a pauper, he deserved better, I was left with nothing but thousands and thousands of medicals bills, and a deceased husband.

The VA is a shrew government agency, when he was rated 100% permanent and totally disabled he was intitled to a 10K life insurance policy that I was not aware of, no one told me, the VA sure won't tell you I found out after the two year statue of limitations and he never received it. I surely could have used that for his funeral. It's things like this that emotionally hurt a veterans family, a surviving spouse, and children. He was in a rental casket, cremated, I wanted

him to be buried, he has no headstone, it has broken my heart and my children's.

I am asking this court to be fair and acknowledge this grisious act of failing to diagnose and treat my beloved husband, it resulted in a wrongful death. His death has devastated me, my children and my beloved grandchildren who will never grow up to know him. What ever this court would choose to compensate me with I would accept with a grateful heart, his death has left me numb.

Thank you for your time and consideration of my claim, I will wait patiently on your decision.

Sincerely,

A handwritten signature in cursive script, appearing to read "Cynthia A. Vann".

Cynthia A. Vann

1701 E. Hebron Pkwy #2101

Carrollton, Texas 75010

Enclosed: 5 pages of Dallas VA Hospital medical records proving they knew he had an Agent Orange illness, heart disease.

1 page showing Cardiac Arrest at time of death at UT Southwestern

3 pages of heart disease information

Radiology Reports

Printed On Jan 12, 2015

using the TeraRecon workstation.

Clinical History: 61-year-old male with shortness of breath.

Findings: Given motion artifact, there is some loss of sensitivity for evaluation of pulmonary emboli but allowing for this limitation, no filling defects identified centrally or in the segmental or subsegmental pulmonary arteries.

Evaluation of the lung windows demonstrates bilateral small pleural effusions and nonspecific reticular and groundglass densities peripherally in a subpleural location bilaterally which is nonspecific but may represent atelectasis and/or pulmonary edema. However, infection cannot be ruled out. Centrilobular emphysematous changes and bulla are identified. No dominant pulmonary nodular masses identified. No pneumothorax

Atherosclerotic calcification is noted in the coronary arteries.

No pathologically enlarged axillary lymph nodes. Subcentimeter mediastinal lymph nodes are noted and are mildly more prominent when compared to prior study suggesting reactive changes. No significant pericardial effusion. Evaluation of the upper abdomen reveals no intervals significant adverse change. Evaluation of the bones reveals degenerative changes including Schmorl's nodes. There is depression of the superior endplate of T7, T11, T12, L1, and L3 vertebral bodies. There is also depression of the superior endplate and inferior endplate of T9. This suggests multiple compression fractures, age-indeterminate. No aggressive lytic or blastic lesions identified.

Impression:

1. Limited evaluation secondary to motion artifact but allowing for this limitation, there is no filling defect identified centrally or in the segmental or subsegmental pulmonary arteries.
2. Bilateral small pleural effusions with bilateral reticular and ground glass densities peripherally in a subpleural location which is nonspecific but may represent atelectasis and/or pulmonary edema. A component of infection cannot be ruled out.
3. Subcentimeter pulmonary lymph nodes which are slightly more prominent compared to prior study which may be reactive.

PATIENT NAME AND ADDRESS (Mechanical imprinting, if available)

VANN, PHILIP DONOVAN
1701 E. HEBRON PKWY
APT 2101
CARROLLTON, TEXAS 75010

VISTA Electronic Medical Documentation

Printed at NORTH TEXAS HCS

Discharge Summaries

Printed On Feb 13, 2013

ACTIVITY: fall precautions

FOLLOW UP:

MS clinic on 8/7/06

DAL-AC-PRIMARY CARE WITH Anthony Jung in 1 month

MEDICATIONS:

Prednisone taper: 200 mg po daily x 2 days, then 150 mg po daily x 2 days, then 100 mg po daily x 2 days, then 90 mg po daily x 1 day, then 80 mg po daily x 1 day, then 70 mg po daily x 1 day, then 60 mg po daily x 1 day, then 50 mg po daily x 1 day, then 40 mg po daily x 1 day, then 30 mg po daily x 1 day, then 20 mg po daily x 1 day, then 10 mg po daily x 1 day, then stop

Omeprazole 40 mg po daily
Caclium/vitamin D 2 tabs po bid
Simavstatin 40 mg po daily
Compazine 10 mg po q6 hrs prn n/v

/es/ JOSEPHR BERGER, M.D.
RESIDENT PHYSICIAN
Signed: 07/28/2006 19:12

/es/ STUART J. SPECHLER, MD
STAFF PHYSICIAN
Cosigned: 07/30/2006 06:54

LOCAL TITLE: Discharge Summary

ADMIN DATE: JUN 05, 2006

DISCH. DATE:

STANDARD TITLE: DISCHARGE SUMMARY

DICT DATE: JUN 12, 2006@09:15

ENTRY DATE: JUN 12, 2006@09:16:05

DICTATED BY: SMITH,ALLISON L

ATTENDING: ONEAL,BARRY L

URGENCY: routine

STATUS: COMPLETED

Admission Date: 06/05/06

Discharge Date: 06/12/06

Attending: Barry Oneal, MD
Resident: Usman Baber, MD
Intern: Allison Smith, MD

Primary diagnosis: Multiple sclerosis

Comorbid conditions: Steroid-induced hyperglycemia

Hyperlipidemia *ATHEROSCLEROSIS*
Agent orange exposure

PATIENT NAME AND ADDRESS (Mechanical imprinting, if available)

VISTA Electronic Medical Documentation

VANN, PHILIP DONOVAN
1701 E. HEBRON PKWY
APT 2101
CARROLLTON, TEXAS 75010

Printed at NORTH TEXAS HCS

Exam date 2-25-2013

Radiology Reports

Printed On Jan 12, 2015

phase of imaging and coronal reconstructions were generated by the technologist. Subsequent 5 mm transaxial images were obtained from the lung apices to the bases. The study was also reviewed using the TeraRecon workstation.

Findings:

No filling defects are identified within the pulmonary arteries to suggest pulmonary embolism. Multifocal atherosclerotic calcifications of the coronary arteries are noted. No significant pericardial effusion. Atherosclerotic calcifications are noted within the thoracic and visualized abdominal aorta, including its major branch vessels. Incidental note is made of a common origin of the brachiocephalic and left common carotid arteries.

Evaluation of the lung windows again demonstrates severe centrilobular emphysema with bullous changes in the lung apices. Stable peripheral reticular and groundglass densities likely represent scarring. No dominant pulmonary nodule or mass. No significant pleural effusion or pleural thickening noted. The trachea and right and left main bronchi and segmental bronchi are normally patent.

Evaluation of the mediastinal windows demonstrates stable subcentimeter mediastinal and hilar lymph nodes. No pathologically enlarged mediastinal, hilar, or axillary lymph nodes evident. Bilateral gynecomastia.

Evaluation of the imaged portions of the upper abdomen demonstrates no acute findings. Stable 8 mm lesion within the right hepatic lobe (series 7, image 50). The liver appears diffusely fatty infiltrated. The visualized spleen and adrenals are unremarkable. Fatty infiltration of the pancreas. Again seen is a 4.2 cm simple cyst within the mid left kidney. The visualized right kidney is unremarkable.

Review of the bone windows demonstrates no acute or aggressive, osteolytic, destructive bone lesion. Bones appear osteopenic. Multiple thoracic compression fracture deformities are again noted.

Impression:

1. No CT evidence of pulmonary thromboembolism.
2. Severe centrilobular emphysema with bullous changes in the lung apices.

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)

VANN, PHILIP DONOVAN
1701 E. HEBRON PKWY
APT 2101
CARROLLTON, TEXAS 75010

VISTA Electronic Medical Documentation

Printed at NORTH TEXAS HCS

Dallas VA Hospital

PROCEDURE : CT CHEST ANGIOGRAPHY W/O&W I

PAGE NUMBER : 1

DATE PRINTED : JUL 26, 2012 10:31

(Case 8878 COMPLETE) CT CHEST ANGIOGRAPHY W/O&W INCLUD(CT Detailed) CPT:71275

Contrast Media : Non-ionic Iodinated

Reason for Study: SOB,

Clinical History:

Suspected PE, d-dimer is high.

Report:

CTA for suspected Pulmonary Embolism

Comparison exam(s): CT chest from 3/30/2010 and 7/11/2006

Technique: Following bolus administration of intravenous contrast using Omnipaque 350 total volume 130 ml, contiguous thin section transaxial images were obtained during the pulmonary arterial phase of imaging and coronal reconstructions were generated by the technologist. Subsequent 5 mm transaxial images were obtained from the lung apices to the bases. The study was also reviewed using the TeraRecon workstation.

Clinical History: 61-year-old male with shortness of breath.

Findings: Given motion artifact, there is some loss of sensitivity for evaluation of pulmonary emboli but allowing for this limitation, no filling defects identified centrally or in the segmental or subsegmental pulmonary arteries.

Evaluation of the lung windows demonstrates bilateral small pleural effusions and nonspecific reticular and groundglass densities peripherally in a subpleural location bilaterally which is nonspecific but may represent atelectasis and/or pulmonary edema. However, infection cannot be ruled out. Centrilobular emphysematous changes and bulla are identified. No dominant pulmonary nodular masses identified. No pneumothorax

*Atherosclerotic calcification is noted in the coronary arteries. No pathologically enlarged axillary lymph nodes. Subcentimeter mediastinal

dictated FEB 3, 2012

VERIFYING STAFF: RICE, GREGG DOUGLAS

typed FEB 3, 2012 15:12

/FEB 3, 2012 15:57

PATIENT NAME : VANN, PHILIP DONOVAN

SOC SEC NUM : [REDACTED]

DATE OF BIRTH: 02-22-1950

AGE : 62

SEX : MALE

CASE NUMBER: 8878

EXAM DATE : FEB 3, 2012 11:40

WARD/CLINIC: 5AMICU

PHYSICIAN : CHOUSAND, MAXI T

RADIOLOGY REPORT

DVAMC NORTH TEXAS HCS

SF 519a

Procedures by Rosechelle Mary Ruggiero, MD at 10/12/14 1524

Author: Rosechelle Mary Ruggiero, MD Service: Pulmonary Author Type: Physician
 Filed: 10/12/14 1549 Note Time: 10/12/14 1524 Status: Signed
 Editor: Rosechelle Mary Ruggiero, MD (Physician)
 Related Notes: Related Note by Andrew Ryan Tomlinson, MD (Fellow) filed at 10/12/14 1528
 I was present for procedure above.

Rosechelle M. Ruggiero

Progress Notes by James Eugene Graff, RN at 10/12/14 1433

Author: James Eugene Graff, RN Service: (none) Author Type: Registered Nurse
 Filed: 10/12/14 1535 Note Time: 10/12/14 1433 Status: Signed
 Editor: James Eugene Graff, RN (Registered Nurse)
 Called to room for Code Blue, pt lost pulse after losing pulse ox reading. ACLS begun.

Procedures by Andrew Ryan Tomlinson, MD at 10/12/14 1524

date of death

Author: Andrew Ryan Tomlinson, MD Service: Pulmonary Author Type: Fellow
 Filed: 10/12/14 1525 Note Time: 10/12/14 1524 Status: Signed
 Editor: Andrew Ryan Tomlinson, MD (Fellow)
 Related Notes: Cosigned by Rosechelle Mary Ruggiero, MD (Physician) filed at 10/12/14 1549

Pre-procedure Diagnoses
 1. Cardiac arrest [427.5]

Post procedure Diagnoses:
 1. Cardiac arrest [427.5]

Procedures
 1. ARTERIAL CATHETERIZATION/CANNULATION, MONITORING/TRANSFUSION (SEP PROC); PERCUTANEOUS [36620 (CPT®)]

Procedure Note - Arterial Line (Right femoral):

Providers - Andrew Tomlinson, MD (fellow); Ray Hwang, MD (resident)
 Medicines - none

Printed on 11/4/2014 3:12 PM

The FASEB Journal

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doi: 10.1096/fj.14-0402ufm

April 2014

The FASEB Journal vol. 28 no. 4 1531-1533

Agent Orange and Heart Disease: Is There a Connection?

Jerome Lowenstein¹

 Author Affiliations

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Spraying Agent Orange: A photo provided by the U.S. Air Force shows four "Ranch Hand" C-123 aircraft spraying liquid defoliant on a suspected Viet Cong position in South Vietnam, September 1965. The four specially equipped planes covered a 1000-foot-wide swath in each pass over the dense vegetation (AP Photo/U.S. Air Force).

In August 2011, a patient asked if I would complete a form that he had received from the Veterans Administration. It was an Agent Orange Fast Track Claim for Ischemic Heart Disease. I completed the form, which requested that I provide evidence that my patient, who had served in Vietnam and described his exposure to Agent Orange, had arteriosclerotic heart disease. That was not difficult, as he had already, at the age of 56, undergone a **coronary artery angioplasty and stent**. I was not aware of a connection between Agent Orange and coronary heart disease.

I learned in May 2013 that compensation was awarded to my patient. I knew that the Veterans Administration, over several years, had recognized Agent Orange exposure as a possible cause of a variety of conditions, predominantly skin disorders and a number of tumors and leukemia. In its most recent deliberations, the Veterans Administration added Parkinson's disease and perhaps most importantly, ischemic (**atherosclerotic heart disease**). This led me to look into the tangled history of Agent Orange during the Vietnam conflict between 1962 and 1971.

Sifting through the evidence

The United States sprayed Agent Orange, a herbicide, in a highly concentrated form as a defoliant in Vietnam beginning in 1962. Agent Orange was known to contain the contaminant TCDD or 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (dioxin), which is regarded as a highly toxic chemical agent in animals and man (1).

During the decade between 1980 and 1990, as veterans' groups were asserting that they were suffering effects of toxicity related to exposure ...

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heart.org

Heart and blood vessel disease — also called **heart disease** — includes numerous problems, many of which are related to a process called **atherosclerosis**. Atherosclerosis is a condition that develops when a substance called plaque builds up in the walls of the arteries. This buildup narrows the arteries, making it harder for blood to flow through. If a blood clot forms, it can stop the blood flow. This can cause a heart attack or stroke.

A **heart attack** occurs when the blood flow to a part of the heart is blocked by a blood clot. If this clot cuts off the blood flow completely, the part of the heart muscle supplied by that artery begins to die. Most people survive their first heart attack and return to their normal lives to enjoy many more years of productive activity. But having a heart attack does mean you have to make some changes. The doctor will advise you of **medications** and **lifestyle changes** according to how badly the heart was damaged and what degree of heart disease caused the heart attack. **Learn more at our Heart Attack website.**

An **ischemic stroke** (the most common type) happens when a blood vessel that feeds the brain gets blocked, usually from a blood clot. When the blood supply to a part of the brain is shut off, brain cells will die. The result will be the inability to carry out some of the previous functions as before like walking or talking. A **hemorrhagic stroke** occurs when a blood vessel within the brain bursts. The most likely cause is uncontrolled hypertension (blood pressure).

Some effects of stroke are permanent if too many brain





5/1/17



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Today on AOL

Cindy Vann (cindyvann@verizon.net)

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Sent from my iPhone

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10:09 AM heart.org 85%
Other Types of Cardiovascular Disease

Heart failure: This doesn't mean that the heart stops beating. Heart failure, sometimes called congestive heart failure, means the heart isn't pumping blood as well as it should. The heart keeps working, but the body's need for blood and oxygen isn't being met. Heart failure can get worse if it's not treated. If your loved one has heart failure, it's very important to follow the doctor's orders. Learn more about [heart failure](#).

Arrhythmia: This is an abnormal rhythm of the heart. There are various types of arrhythmias. The heart can beat too slow, too fast or irregularly. Bradycardia is when the [heart rate](#) is less than 60 beats per minute. Tachycardia is when the heart rate is more than 100 beats per minute. An arrhythmia can affect how well the heart works. The heart may not be able to pump enough blood to meet the body's needs. Learn more about [arrhythmia](#).

Heart valve problems: When heart valves don't open enough to allow the blood to flow through as it should, it's called [stenosis](#). When the heart valves don't close properly and allow blood to leak through, it's called [regurgitation](#). When the valve leaflets bulge or prolapse back into the upper chamber, it's a condition called [prolapse](#). Discover more about the [roles your heart valves play in healthy circulation](#) and learn more about [heart valve disease](#).

Cardiovascular Disease	Treatment
Heart Valve Problems	Medications Heart Valve Surgery

